UNITED STATES DISTRICT COURT
FOR THE MIDDLE DISTRICT OF TENNESSEE
NORTHEASTERN DIVISION

DAVID CHERRY, personal representative of the estate of PAMELA CHERRY, deceased, Plaintiff, vs. MACON HOSPITAL, INC. d/b/a MACON COUNTY GENERAL HOSPITAL and HANNA C. ILIA, M.D., Defendants.	)	CASE NO. 2:12-cv-00043

DEFENDANT HANNA C. ILIA, M.D.'s EXPERT DISCLOSURES

Defendant Hanna C. Ilia, M.D. provides herein his Rule 26(a)(2) Disclosures, which also serve as supplemental responses to Interrogatory no. 8 of Plaintiff's First Set of Interrogatories to Defendant Hanna C. Ilia, M.D. Through counsel, Dr. Ilia herein advises Plaintiff that he reserves the right to call the following individuals as Rule 26 expert witnesses at the trial of this matter:

I. EXPERT WITNESS IDENTITIES

1. Alan E. Jones, M.D., FACEP
University of Mississippi Medical Center
Department of Emergency Medicine
2500 North State Street
Jackson, Mississippi 39216

2. William B. Hillegass, Jr., M.D.
University of Alabama Hospital
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3. Sanjeev Saxena, M.D., F.A.C.C.
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Background Risk (prior probability of Coronary Artery Disease (CAD))

In the emergency room or office assessment of patients with chest pain of possible cardiac etiology, the first task is to assess the baseline risk or prior probability of prognostically significant CAD. It is my opinion that this patient, Pamela Cherry, did not have a history of coronary artery disease. She apparently did not report a history of coronary artery disease to Dr. Ilia or the nurses at Macon County General Hospital (MCGH). Her past medical records indicate no prior history of or clinical diagnosis of coronary disease.

It is my opinion that Pamela Cherry also did not have a family history of coronary artery disease. A patient has a family history of coronary artery disease, and is therefore at higher risk of cardiovascular events, if a family member experiences a cardiovascular event before the age of 55 (for males) or before the age of 65 (for females). Cardiovascular events after these ages do not discriminate the patient from the background population risk. This patient did not have a family history of coronary artery disease that would have placed her at a higher risk of a cardiovascular event.

She stated that she was a smoker at one pack per day. (As well documented in her past medical records, her primary physicians had counseled her on numerous occasions in the preceding years to cease smoking as a lifestyle modification to improve her own health outlook.)

She had hyperlipidemia. Past records indicated this was relatively mild dyslipidemia and at times was well-controlled on 40 mg of simvastatin. She also stated that she was taking simvastatin, a lipid-altering agent, at the time of presentation to MCGH. When the patient informed the nurse and doctor at MCGH that she was taking simvastatin, they could properly assume the patient was being truthful. When an individual is treated with a statin for hyperlipidemia, this risk factor can be assumed to be mitigated as a risk factor for heart disease with substantially lower risks of death, myocardial infarction, and death from myocardial infarction.

Medical records from her pharmacy indicate that this patient was not taking simvastatin as prescribed. Her thirty (30) day supply was last filled on February 23, 2011, more than three months prior to the time Dr. Ilia saw her. Once a patient stops taking simvastatin, it loses its protective effect. If the patient had been taking simvastatin as directed, her probability of having a heart attack would have been substantially lower. Even if she had still suffered a heart attack, her likelihood of death would have been lessened if she had been taking simvastatin as directed.

Overall, given this risk factor profile, based on the Framingham risk calculator with her most recently available lipid profile and other risk factors, her 10 year risk of myocardial infarction would have been judged to be 3%. This documents a very low background risk of myocardial infarction. The PROCAM model would estimate a 10 year risk of death or nonfatal MI at 2.1%.

The most frequently used and validated risk prediction instruments yield very low prior probabilities for cardiovascular death or nonfatal MI in individuals with Ms. Cherry's background risk profile. Whether explicitly calculated or most often implicitly estimated by an experienced clinician, estimation of this background risk (prior probability of prognostically significant CAD) is one of the most powerful determinants of the likelihood that the chest pain presentation reflects an acute coronary syndrome (ACS). Hence, the evaluating clinician would be appropriately assuming a very low prior probability of prognostically significant coronary disease and acute coronary syndrome in patients like Ms. Cherry.

Presentation and Evaluation

Given the very low prior probability of prognostically significant CAD or ACS in patients like Ms. Cherry, fairly definitive evidence from the specific presentation and evaluation would be necessary to significantly modify the final assessment (posterior probability) of the likelihood of CAD or ACS in the usual appropriate medical decision-making "Bayesian" process.

Presentation EKG. The EKG from May 30, 2011 was non-diagnostic in my opinion. The "ST depressions" that are described in the Plaintiff's Expert Disclosure appear to be only one-half (½) millimeter. Moreover, they are present only in two anterolateral leads V5 and V6, and do not appear in inferior leads. This degree and pattern of ST deviation is relatively non-specific. These leads frequently exhibit repolarization abnormalities with downsloping ST depression in a variety of medical conditions. It should be noted that the magnitude of the ST changes were insufficient for the GE Healthcare computerized EKG interpretation algorithm to detect and report anterior or anterolateral ST segment depressions. Many risk prediction tools for diagnosis of acute coronary syndromes in the emergency room patient have found a threshold of >1 mm ST depression as providing the best trade-off between sensitivity and specificity for diagnosing an appreciable likelihood of an acute coronary syndrome. Therefore, in and of itself, the presentation EKG is not specific for diagnosis of an acute coronary syndrome. Overall, the non-specific, non-diagnostic, and relatively minimal EKG changes would not mandate admission of the patient to the hospital without other compelling clinical evidence.

(The GE Healthcare algorithm did read "possible anterior infarct – age undetermined" due to poor R-wave progression. Poor R-wave progression is a notoriously nonspecific and inaccurate criterion for new or old myocardial infarction. It is mainly associated with remote anterior MI but is appropriately discounted in the absence of ST elevation and/or no history of remote MI. Given the finding of right coronary artery occlusion or severe stenosis with near normal left coronaries, as found at the catheterization at Vanderbilt University Medical Center in Ms. Cherry, one would have typically expected to find ST elevation or depressions in limb leads II, III and AVF which was not present on the EKG.)

Additionally, the May 30, 2011 EKG was not sufficiently suspicious in the setting of the entire clinical picture to require tracking down previous EKG's to make an informed clinical decision. Nevertheless, if previous EKG's had been obtained and reviewed, it would have been unlikely to have altered the course of action. The patient's previous records include prior EKG's from (*a*) March 2009, at which time the patient underwent shoulder surgery, and from (*b*) May 2010, at which time she presented to an emergency room complaining of back and chest pain. Three of these four EKG's would be deemed "abnormal" with non-specific ST-T changes. Review of these previous EKGs would have established a "not normal" baseline, and the sensitivity and specificity of the changes on the May 30, 2011 EKG would have been further diminished. Hence, availability of old EKGs would have only further diminished the diagnostic utility of the presentation EKG.

Presentation Symptoms and Physical Findings. There were atypical symptoms, physical findings, and response to initial therapies that increased the difficulty in evaluating the patient on presentation in the emergency department. This patient had significant sunburn which both the nurse and Dr. Ilia describe as corresponding with the location of pain complaints in her back, chest, neck and jaw. Dr. Ilia described her sensitive reaction to the touch of the stethoscope upon her chest, and he noted that her back appeared to be hurting the most. These observations

suggested a cause other than a cardiac event and, instead, indicated that the patient's pain was most likely attributable to sunburn and/or musculoskeletal causes. The patient also received no pain relief from nitroglycerin but did receive detectible improvement from Toradol. Toradol is a NSAID drug which can be administered intravenously for musculoskeletal discomfort. In fact NSAIDs are a first line and effective treatment for severe sun burn discomfort within 24 hours of excessive sun exposure. This was further indication that the patient's pain complaints were musculoskeletal-related and/or most likely sunburn-related, not cardiac-related. In the context of atypical pain symptoms and physical findings for ACS, non-specific EKG findings have been shown to have even lower diagnostic accuracy for ACS.

Troponin. The Troponin-I assay is a sensitive assay. Studies have shown that the diagnostic accuracy of the sensitive Troponin-I assay is highest, with an AUC of 0.95 in patients presenting within three (3) hours after onset of chest pain. This value does increase, but only very slightly to 0.96, in patients presenting within six (6) or (12) hours after onset. Troponin-I assays have a highly negative predictive value when measured within three (3) hours of the onset of chest pain. Dr. Ilia was informed that Ms. Cherry's symptoms began early in the afternoon while washing a car consistent with testimony from the family. Thus the Troponin test would be expected to have very high sensitivity for an ACS.

The patient's troponin level was reported as 0.06 which was within the normal reference range of 0.00 – 0.10 used by the Macon County General Hospital Laboratory. For this troponin-I assay, results less than 0.10 are not sufficiently sensitive or specific for acute coronary syndrome or myocardial infarction to provide clinically meaningful incremental risk stratification. Only patients with results of at least 0.10 are at sufficiently increased risk for adverse outcomes to significantly modify the probability of acute coronary syndrome to alter clinical decision making. Hospitals typically develop their own reference intervals and decision limits, and it was appropriate for Dr. Ilia to follow the appropriate reference range adopted here.

Moreover, while other hospitals using other assays may include "indeterminate" values within their troponin reference ranges, such values are commonly referred to as "Troponinemia" and are generally disregarded in the decision-making process as adding no appreciable incremental value over the clinical presentation in revising the final assessment "posterior probability" of the likelihood of ACS. Even if this patient's troponin value had reached the "indeterminate" range within another assay, such a value would have been non-diagnostic and would not have dictated further or different actions on the part of Dr. Ilia.

Risk Assessment for ACS. Though Dr. Ilia ultimately concluded that the patient's pain complaints were not cardiac-related, his assessment that the risk of ACS was sufficiently low to justify discharge is supported by numerous studies and tools for predicting the likelihood of prognostically significant ACS. If acute coronary syndrome has crossed the mind of the emergency medicine physician, it is within the standard of practice to discharge the patient if there is less than 3 to 5 percent (3-5%) probability the patient is experiencing acute coronary syndrome. For example, the Thrombolysis In Myocardial Infarction (TIMI) risk score for this patient's presentation would assess a 4.7% risk at 14 days of death, new or recurrent myocardial infarction, or severe recurrent ischemia requiring urgent revascularization. Other assessment scales developed specifically for patients presenting to emergency departments with chest pain yield even lower risk calculations. Under the H.E.A.R.T. ("History-EKG-Age-Risk factors-Troponin") risk score, this patient's risk score would total 3, equating to a 0.9% risk of death, myocardial infarction, coronary bypass, and/or coronary intervention at 30 days. And although the "Fail-Safe Checklist" employed at Macon County General Hospital was designed as a

reminder of factors that may be useful in risk stratification, this patient's presentation would have resulted in larger number of negative than positive indicators. In more quantitative composite scoring systems such as TIMI or HEART, the sum of these factors in the MCGH checklist would yield a very low likelihood of ACS.

Summary Clinical Judgment Based on Prior Probability and Presentation/Findings/Evaluation

This patient had a very low baseline risk (prior probability) of prognostically significant coronary disease. She also had a low-probability presentation for acute coronary syndrome given the atypical symptoms, physical findings, non-specific EKG, normal troponin, response to therapies, and clinical course. The appropriateness of Dr. Ilia's clinical judgment in discharging her is well supported by the formal risk stratification research and tools that have helped define the standard of care in this field of emergency cardiovascular medicine. Further, in individuals like Ms. Cherry with very low probabilities of ACS by TIMI Risk Score or HEART Score, the literature (including meta-analysis of all relevant studies and consensus expert opinion statements such as "Up-To-Date" and societal practice guidelines) explicitly state that the evaluating physician's overall clinical judgment as to the probability of ACS is the necessary standard of care. Based upon Dr. Ilia's assessments, the consistency between the patient's pain complaints with sunburn pattern, her normal vital signs, monitor readings, reaction to medications, EKG and lab reports, and her entire clinical picture, it was appropriate and within the standard of acceptable professional practice for Dr. Ilia to discharge Pamela Cherry on May 30, 2011.

Other Facts

EMS records from Celina Fire Department EMS state that the patient was without resuscitative measures for approximately 20 minutes. But family witnesses stated that CPR was administered after the patient initially collapsed. I can comment further when I am provided the transcripts of the EMS providers' depositions. I can comment now that the prompt if not immediate administration of CPR and rescue breathing is critical and that, in the absence of timely resuscitative efforts, irreversible damage including brain anoxia can result within less than 10 minutes. Current clinical mortality is essentially 100% if >10 min elapse before starting CPR.

The materials in the Plaintiff's Expert Disclosure states that this patient's subsequent course, in the most likely scenario, was that she had a clot form in her right coronary artery which initially was not occlusive. The Disclosure states that by the next day, the clot had progressed to complete occlusion. Records from Vanderbilt Medical Center do identify an acute proximal occlusion with thrombus in the right coronary artery as the culprit lesion. The issue is whether admitting the patient to Macon County General Hospital on the evening of May 30, 2011 would have altered this outcome. It is highly unlikely that she would have been transferred to an outside facility based upon her low prior probability of CAD and low probability of ACS clinical presentation on 5/30/2011. If the patient had a restful night in the hospital, as it appears she did at her in-law's home, her monitoring and cardiac markers would likely have remained within normal limits until the onset of occlusion at approximately 6:00 a.m. Even assuming continued telemetry and repeated cardiac enzymes by medical staff, no interventional care would

likely have been indicated or provided before her arrest. In other words, the patient would have nevertheless suffered a heart attack and cardiac arrest, but in the hospital as opposed to her in-law's home. Assuming the medical staff would have been able to restore a sinus rhythm, an EKG would have been done and would likely be markedly abnormal. She would have then most likely been treated with thrombolytic drugs in a non-PCI facility such as MCGH and then transferred to a tertiary facility. Therefore, in my opinion, even if the patient had been admitted to MCGH on the evening of May 30, 2011, subsequently suffering an arrest in the hospital, her chance of a more favorable outcome would require successful resuscitation from most likely VT/VF arrest(s), thrombolysis, and other life support (most likely including a ventilator) followed by transfer to tertiary facility where PCI could be provided if indicated at that juncture largely depending on neurologic status after the foregoing events.

It is my opinion to a reasonable degree of medical certainty that Dr. Ilia did not cause Pamela Cherry's death. Similarly, her death was not the result of any act or omission on the part of the Macon County General Hospital nurses or staff. It is also my opinion that there is no evidence to support the assertion that Mrs. Cherry experienced any mental or physical suffering between the time of discharge from the emergency department on May 30, 2011 until the time of her death. I am familiar with the recognized standard of acceptable professional practice applicable to emergency medicine physicians and nurses practicing in Lafayette, Tennessee or similar communities as those standards existed in 2011. In my opinion, Dr. Ilia and the medical staff at Macon County General Hospital complied with the applicable standards of care. These are my opinions within a reasonable degree of medical certainty. I can expand upon my opinions upon receipt of additional materials and/or at a deposition if requested.

I have never given deposition testimony or trial testimony. My compensation in this matter is based upon an hourly rate of $250.00 an hour, similar to one's earnings from the clinical practice of cardiology.

Sincerely,

William Hillegass, M.D.